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# Peste Des Petits Ruminants

**Seema Agarwal**
College of Veterinary and Animal Sciences, G.B. Pant University of Agriculture and Technology, Pantnagar-263 145 Udham Singh Nagar (Uttarakhand) INDIA

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Peste des petits ruminants (PPR) is an acute or subacute viral disease of goats and sheep characterized by

- Fever
- Necrotic stomatitis
- Gastroenteritis
- pneumonia, and sometimes death.

Goats and sheep appear to be equally susceptible to the virus, but goats exhibit more severe clinical disease. The virus also affects several wild small ruminant species. Cattle, buffalo, and pigs are only subclinically infected. People are not at risk.

In 1942 it was first time reported in Ivory Coast in West Africa by Gargadennec & Lalanne, and subsequently in sub- Saharan Africa by Senegal. Shaila et al., reported in 1996 in the Arabian Peninsula, the Middle East and the Indian subcontinent. In 1962 PPR virus (PPRV) first isolated in sheep cell culture. The first confirmed outbreak in India in sheep in village Arasur in Villapuram district of Tamil Nadu was reported by Shaila et al., in1989. In North India first reported from HP in1996.Disease can affect some wild ungulates, but there is very limited information on species susceptibility and the occurrence of disease Two severe outbreaks were reported in Saudi Arabia in 2002. Disease had severely affected Buffalo in India during 1995. White-tailed deer (Odocoileus virginianus) infected experimentally. Captive Nubian ibex, Laristan sheep and gemsbok wild ruminants may be important in the epidemiology of this disease but exact role is unknown. Though sheeps and goats are the main host but sometimes also affect cattle & buffaloes, wild ungulates (gazelles, white tailed deer).

**Transmission**

- Transmission is by close contact, and confinement seems to favor outbreaks.
- Secretions and excretions of sick animals are the sources of infection

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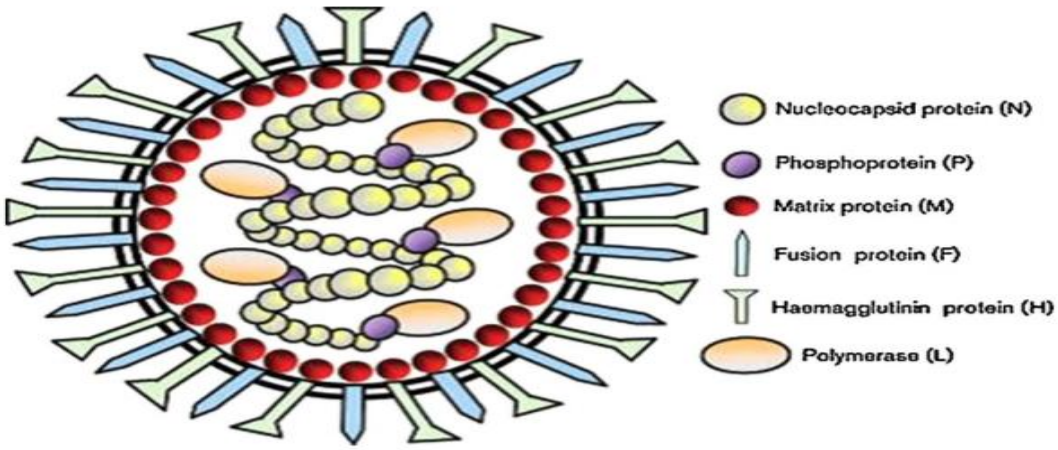
- Several species of gazelle, oryx, and white-tailed deer are fully susceptible; these and other wild small ruminants may play a role in the epidemiology of the disease, but few epidemiologic data are available for PPR in wild small ruminants
- Cattle, buffalo, and pigs can become naturally or experimentally infected with PPR virus, but these species are dead-end hosts, because they do not exhibit any clinical disease and do not transmit the virus to other in-contact animals of any species

**Etiology:**




It is caused by a single stranded non-segmented virus belonging to the genus Morbillivirus, family Paramyxoviridae. The virus exists as a single serotype but, by means of nucleic acid sequencing, it can be differentiated into four lineages (1–4). It is antigenically similar to rinderpest virus, measles virus and canine distemper virus. The virus remains stable at the pH of 4-10. Until recently, this virus was named simply Peste des petits ruminants virus (PPRV); the official name of this virus was changed in 2016 to Small ruminant morbillivirus (SRM).However, it is still commonly known as PPRV by people working in the field.

**Epidemiology:**

**SMALL RUMINANT AT RISK BY INFECTED REGION**

| | |
|---|---|
| Central Asia | 43,118,821 |
| Near East | 171,997,500 |
| Far East | 647,518,989 |
| Africa | 264,275,400 |
| **Total population at risk** | **1,126,910,710** |
| Global small ruminant population | 1,801,434,416 |
| **Rate of the global domestic small ruminant population is at risk** | **62.5%** |

*Souce:* FAOSTAT

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PPR is one of the most economically important animal diseases of small ruminants. Outbreaks tend to be associated with contact of immuno-naïve animals with animals from endemic areas. The disease is prevalent in West Africa and the Middle East.

- Morbidity rate in susceptible populations can reach 90–100%.
- Mortality rates vary among susceptible animals but can reach 50–100% in more severe instances.
- Both morbidity and mortality rates are lower in endemic areas and in adult animals when compared to young
- Cattle develop inapparent infections and do not transmit disease
- Pigs have been reported as being susceptible and transmitting the virus under laboratory conditions but so far not in the field
- May be associated with limited disease events in camels, but they do not appear to transmit the virus. The virus is secreted in tears, nasal discharge, secretions from coughing, and in the faeces of infected animals. It particularly affects young ones of four months to one year of age. The virus has been circulating in parts of sub-Saharan Africa for several decades and in the Middle East and southern Asia since 1993, although the first description of the virus in India dates to 1987.

To study the genetic relationship between isolates of distinct geographical origin, a selected region of the fusion (F) protein gene of the viruses was amplified using RT/PCR and the resulting DNA product sequenced for phylogenetic analysis.

- Viruses from 27 outbreaks in Asian and Middle Eastern countries, reported between 1993 and 2000, and two recent outbreaks from the African continent were compared with the prototype African strain. Of the four known lineages of PPR virus, lineage 1 and 2 viruses have been found exclusively in West Africa.
- Virus from an outbreak in Burkina Faso in 1999 fell into the lineage 1 group. Viruses of lineage 3 have been found in East Africa, where an outbreak in Ethiopia in 1996 was of this type, and also in Arabia and in southern India. However, there have been no further isolations of lineage 3 virus from India since the one reported in 1992 from Tamil Nadu.
- A virus of this lineage was found circulating in Yemen in 2001. In the past 8 years virus exclusively of the fourth lineage has spread across the Middle East and the Asian

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subcontinent, reaching east as far as Nepal and Bangladesh. This virus lineage was also reported from Kuwait in 1999.

- The geographical source of the new lineage 4 virus is unknown although it is most closely related to African lineage 1. The possibility that its earlier presence in northern India was masked by the circulation of Rinderpest virus, a related virus of cattle, is considered unlikely.

**Pathogenesis:**

PPR virus penetrates the retropharyngeal mucosa, sets up a viremia and specifically damages the alimentary, respiratory and lymphoid systems. Infected cells undergo necrosis, and in the respiratory system, also proliferation. Death may occur from severe diarrhoea and dehydration, before respiratory lesions become severe, or is hastened by concurrent diseases such as pneumonic pasteurellosis, coccidiosis or coliform enteritis. Lymphoid necrosis is not as marked as in rinderpest and the possibility of immunosuppression has not been investigated. Most sheep and some adult goats recover.

**Necropsy Findings**

The carcass is severely dehydrated, the hindquarters are soiled with fluid feces, and crusts of exudate are present around eyes, nose and lips. Discrete or extensive areas of erosion, necrosis, and ulceration are present in the oral mucosa, pharynx, and upper esophagus and may extend to the abomasum and distal small intestine. Hemorrhagic ulceration is marked in the ileocecal region, colon and rectum where they produce typical 'zebra stripes'. Regional lymph nodes are enlarged and wet and the spleen may be enlarged. Severe lesions are often present throughout the respiratory tract. A mucopurulent exudate extends from the nasal opening to the larynx whereas the trachea and bronchi may be hyperemic and contain froth due to pulmonary congestion and edema. Interstitial pneumonia is usually present. Grossly, the pneumonia is diffuse or more commonly, antero-ventral or apical. With bacterial complications, there will be purulent or fibrinous bronchopneumonia and pleuritis. Microscopic lesions in the alimentary tract are similar to those in rinderpest but are often more severe. In the early stages, syncytial cells are present in the oral mucosa and intracytoplasmic eosinophilic inclusion bodies in intestinal crypt epithelium. The respiratory tract shows proliferative rhino tracheitis, bronchitis, bronchiolitis, proliferation of type II pneumocytes, and formation of huge syncytial giant cells. Intracytoplasmic and intranuclear

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inclusion bodies are common in these cells. In a recent immunohistochemical study of natural PPR pneumonias in goats, viral antigens were found most frequently in the cytoplasm and rarely in the nucleus of lower respiratory epithelial cells, type II pneumocytes, syncytial cells and alveolar macrophage. Lymphoid organs are depleted of lymphocytes but not usually as marked as in rinderpest. For diagnostic purposes, specimens should be collected from several live animals and should include swabs of conjunctival, nasal and buccal mucosa, as well as whole blood in anticoagulant for virus isolation and other tests. At necropsy, the following specimens should be collected for virology and histopathology:

- Lungs
- Small and large intestines
- Oral mucosa
- Tonsil
- Mesenteric lymph nodes.

**Signs, symptoms and lesions**

The disease can be acute or subacute:

Acute form is seen mainly in goats and is similar to rinderpest in cattle except that severe respiratory distress is a common feature of PPR.

- Signs generally appear 3-6 days after being in contact with an infected animal. A high fever (above 40°C) is accompanied by dullness, sneezing and serous discharge from the eyes and nostrils.
- A day or two later, discrete necrotic lesions develop in the mouth and extend over the entire oral mucosa, forming diphtheric plaques.
- There is profound halitosis and the animal is unable to eat because of a sore mouth and swollen lips.
- Nasal and ocular discharges become mucopurulent and the exudate dries up, matting the eyelids and partially occluding the external nares.
- Diarrhea develops 3-4 d after the onset of fever. It is profuse and feces may be mucus and blood tinged.
- Dyspnea and coughing occur later and the respiratory signs are aggravated when there is secondary bacterial pneumonia.
- Erosions have been described in the vulva and prepuce.

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- Abortions have been reported during outbreaks in India.Death usually occurs within 1 week of the onset of illness.

**Subacute forms** are more common in sheep but they also occur in goats. The signs and lesions are less marked and a few animals may die within 2 weeks, but most recover. Contagious ecthyma may complicate the labial lesions or develop in surviving animals.Characteristic symptoms:-

- Fever (105-107°F)
- Diarrhoea, melena
- Erosive mouth lesions
- Skin eruptions
- Conjunctivitis
- Mortality up to 80%

**Lesions**

- Erosions on oral mucosa
- Bran like deposits on erosions
- On removal haemorrhagic raw surface is present
- Abortion
- Haemorrhage in intestines leading to Zebra markings
- Petechial haemorrhage in bladder, vagina and other mucous membrane
- Congestion of conjunctiva
- Giant cell pneumonia

**Diagnosis**

- A presumptive diagnosis is based on clinical, pathologic, and epidemiologic findings and may be confirmed by viral isolation and identification.
- Historically, simple techniques such as agar-gel immunodiffusion have been used in developing countries for confirmation and reporting purposes. However, PPR virus cross-reacts with rinderpest virus in these tests.
- Virus isolation is a definitive test but is labor intensive, cumbersome, and takes a long time to complete.

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- Currently, antigen capture ELISA and reverse transcription-PCR are the preferred laboratory tests for confirmation of the virus.
- For antibody detection (such as might be needed for epidemiologic surveillance, confirmation of vaccine efficacy, or confirmation of absence of the disease in a population), competitive ELISA and virus neutralization are the OIE-recommended tests.
- The specimens required are lymph nodes, tonsils, spleen, and whole lung for antigen or nucleic acid detection, and serum (from unclotted blood) for antibody detection, The virus neutralization test may also be used to confirm an infection if paired serum samples from a surviving animal yield rising titers of ≥4-fold.
- PPR must be differentiated from other GI infections (eg, GI parasites), respiratory infections (eg, contagious caprine pleuropneumonia), and such other diseases as contagious ecthyma, heartwater, coccidiosis, and mineral poisoning.

**Prevention and control**

**Immunization and Vaccines**

**PPR Vaccine-**

Sungri/96 Strain (The vaccine may be administered in lambs and kids at 4 months of age**.**This vaccine should be administered with a dose of 1 ml by subcutaneous injection at mid neck region. Use precautions including application on clean dry skin, use sterile needles and syringes for every administration.

PPR is a listed disease of the OIE, and thus member states are required to inform the OIE of the occurrence of the disease in their territory. The OIE publishes recommendations for zoo-sanitary conditions and certification of trade in animals and livestock products from countries which are not recognized as having freedom from PPR disease (OIE, 2011). The OIE recommends sanitary prophylaxis (movement control, quarantine of infected premises, with slaughter of infected animals and in-contacts) when the disease appears in previously PPR-free countries. The use of a stamping-out policy, involving slaughter of infected and in-contact animals on infected premises, can lead to a reduced period of time elapsing after the last case of disease has been reported before the country is internationally recognized as free of PPR.

**PPR Global Control and Eradication Strategy**

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Despite the availability of an efficacious and cheap live-attenuated vaccine, the virus has continued to spread. In April 2015, the Food and Agriculture Organization of the United Nations (FAO) and the World Organisation for Animal Health (OIE) launched the PPR Global Control and Eradication Strategy (PPR GCES) with the vision for global eradication by 2030. There is a strong and lasting international consensus to eradicate the disease in order to protect the livelihoods of the world's poorest populations. Vaccination is being taken forward as the key strategy along with epidemiological surveillance to target vaccination efforts and eradicate the disease.

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